Great Lakes Fishery Leadership Institute

Contaminant Issues Relevant to Great Lakes Fisheries

Pennsylvania Sea Grant

Eric Obert
John Cingolani
Sean Rafferty

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Acknowledgement

We would like to give special credit to the EPA and its affiliated Web sites for much of the material presented in this curriculum. In addition, we give credit to information compiled from Agency for Toxic Substances and Disease Registry, Journal of Great Lakes Research, Environmental Health Perspectives, World Resources Institute, Great Lakes Fishery Commission and the Great Lakes Sea Grant Network. A complete list of reference articles is found in the bibliography section.

Contact Information

For further information on contaminants and their effects on aquatic and human health, fish consumption advisories, fish tumors, or avian botulism please contact:

Pennsylvania Sea Grant
Penn State Erie
5091 Station Rd
Erie, PA 16563-0501
Phone: (814) 898-6420
Fax: (814) 898-6461

Introduction

Overview

The purpose of this curriculum is to create awareness of the broad inputs that contaminate sediments can have on aquatic and human health. The curriculum includes detailed information related to types of contaminants, fish consumption advisories, fish tumors, and botulism.

Contaminated sediment in the rivers, lakes, and oceans of the United States has emerged as an ecological and human health issue on a nationwide scale. Contaminated sediment can be associated with both short-and long-term effects on aquatic life. Sediments also serve as a source of bioaccumulative contaminants (e.g. toxic chemicals), which may have a negative effect on ecological and human health even after contaminants are no longer released from point and non-point sources.

The deposition of contaminants in sediment does not mark an end to the problem. Contaminated sediments do not always remain at the bottom of a water body. Anything that stirs up the water, such as a storm or a boat's propeller can resuspend sediment in the water column. Approximately 300 million cubic yards of sediment are dredged in the United States every year in order to deepen harbors and clear shipping lanes. Roughly 3 - 12 million cubic yards of these sediments are severely contaminated and require special, and sometimes costly handling. This resuspension may lead to the increased exposure of the pelagic (open water) fish and benthic species to the contaminants.

The contaminant section of the curriculum relates to the effects estrogenic substances, metals, organics, and fire retardants have on the Great Lakes ecosystems. Estrogenic substances include mercury, polychlorinated biphenyls (PCBs), and dichlorodiphenyl tri-chloroethane (DDT). These substances originate from various sources such as the combustion of fossil fuels from coal-fired power plants, pesticides, and industrial waste.

(Photograph on the left is courtesy of National Park Service, Indiana Dunes National Lake Shore and illustrates Lake Michigan sand dunes with power plant in the background. The photograph on the right is from the Cleveland Public Library and illustrates the 1952 fire of the Cuyahoga River that was caused by pollution.)

Mercury, lead, cadmium, arsenic and chromium are considered to be the most harmful metal contaminants. Metal contaminants are commonly associated with coal-fired power plants, industrial waste, atmospheric deposition, heating of minerals in smelters, and natural sources such as rocks, animals, plants, soil, and volcanic dust.

Polychlorinated biphenyls (PCBs), polycyclic aromatic hydrocarbons (PAHs), and dioxin are categorized as organic contaminants. PCBs come from various sources and were commonly used in electrical transformers, capacitors, and other electrical equipment because of their heat resistance qualities. Most PAHs are a product of the incomplete combustion of fossil fuels in coking operations or from petroleum spills and urban runoff. Dioxins have been associated with herbicides (e.g. agent orange), paper mills, and incineration of plastics.

Fire retardants such as polybrominated biphenyls (PBBs) are manufactured chemicals found in plastics used in a variety of consumer products ranging from computer monitors, televisions, textiles, and plastic foams to make them difficult to burn. Many of these contaminants released into the environment years ago are now banned from use; however, many of these persistent chemicals still remain concentrated in the sediment at the bottom of our rivers, lakes, and oceans.

No single government agency is completely responsible for addressing the problem of contaminated sediments. A variety of laws give federal, state, and tribal agencies authority to address sediment quality issues. Private industry and the public also have roles to play in contaminated sediment prevention. Increasing public environmental awareness of the problem with contaminants is crucial to developing effective remedial actions for contaminated areas.

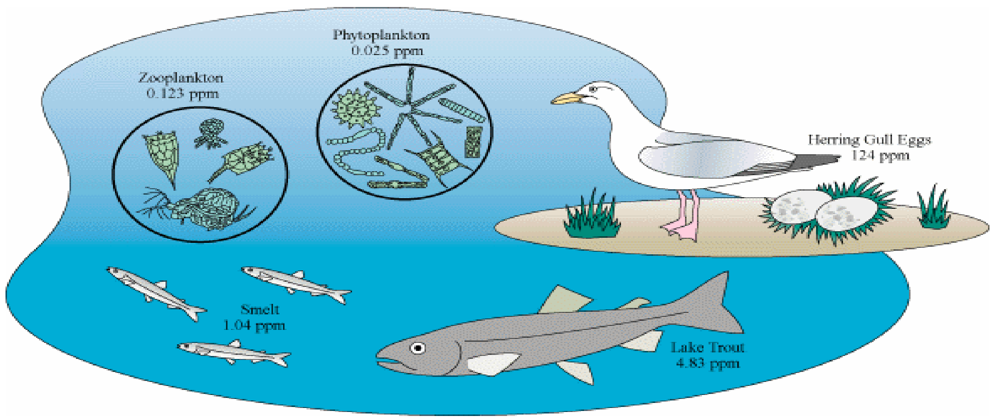
The International Joint Commission (IJC) was formed in 1909, comprised of American and Canadian officials, to assist these governments in finding solutions to the problems facing the waters bordering the United States and Canada, and to manage and protect these waters for the benefit of today's citizens and future generations.

In 1987, the U.S. and Canadian governments signed a protocol promising to report on the progress associated with the improvement of Areas of Concern and requiring the IJC to review Remedial Action Plans (RAPs). Areas of Concern (AOCs) are described as geographic areas, within the Great Lakes Basin, that fail to meet the general or specific objectives of the Great Lakes Water Quality Agreement (GLWQA) where such failure has caused or is likely to cause impairment of beneficial use of the area's ability to support aquatic life.

RAPs are being developed and implemented at the 42 current AOCs. The objective of RAPs is to restore the beneficial uses, as identified in Annex 2 of the Great Lakes Water Quality Agreement (GLWQA). The mechanisms responsible for the loss of ecological integrity in AOCs are identified in the first step of the RAP development process. Plans of action are then designed to restore these areas to levels that meet government and public expectations. The restorative measures use an ecosystem approach which considers not only land, air, and water degradation, but also the loss or restriction of human uses in the Great Lakes Basin.

Potential Effects of Contaminants on Aquatic Life
Contaminated sediments affect benthic (bottom dwelling) aquatic organisms such as worms, crustaceans, and insect larvae in many ways. Some toxic chemicals in sediments kill benthic organisms; thereby, reducing the food available to larger organisms such as fish. Other contaminants in the sediment are taken up by benthic organisms and become concentrated in higher and higher amounts in a process called bioaccumulation. When larger animals feed on these contaminated organisms, the toxins are taken into their bodies, moving up the food chain in increasing concentrations in a process known as biological amplification (Figure 1). As a result, contaminated sediments can affect benthic organisms, fish and shellfish, waterfowl, and freshwater and marine mammals.

Figure 1: Example of Biological Amplification in an Aquatic Ecosystem



(*http://www.homestead.com/concernedcitizens/osf.html*)

Aquatic species that cannot tolerate the toxic contaminants found in sediment die, leading to a reduction of the biodiversity (variety of organisms). Some of these contaminants, acting alone or with other contaminants, are thought to be carcinogens. Fish tumor induction data, tumors in wild fish populations, and the presence of contaminants (e.g. carcinogens) have provided evidence that chemical carcinogenesis is occurring in the Great Lakes and its watersheds. Other problems associated with contaminated sediments, include: fish deformities and reproductive problems.

Consumption Advisories
Fish are a lean low-calorie source of protein and can play an integral part of a healthy diet; however, some of the fish species anglers catch from the Great Lakes and surrounding watersheds could pose health risks because of accumulating contaminants in the tissue of fish and waterfowl. In 1998, fish consumption advisories were issued for more than 2,506 bodies of water in the United States. Possible long-term effects of eating contaminated fish include cancer, learning disabilities, and neurological defects.

Botulism Outbreaks
Botulism has been recognized as a major source of mortality in fish and migratory birds since the early 1900s. Botulism spores are commonly found in the environment but need anaerobic conditions (without oxygen) to develop into a vegetative state capable of

producing toxin. Once in this state the botulism is thought to be concentrated on protein substrates that may include invertebrates (mussels) and aquatic insects. Consumption of these invertebrates results in paralysis and usually death for the fish or bird that is consuming them. There are various types of botulism; however, type E botulism has been mainly restricted to fish-eating birds in the Great Lakes. A recent outbreak of type E botulism on Lake Erie has resulted in the death of thousands of gulls, loons, mergansers, long-tailed ducks, and many benthic fish, including species of concern such as the Lake Sturgeon.

Contaminants
Estrogenic Substances
Background Information
Environmental estrogens are chemical compounds that have serious effects on sexual development. These endocrine-disrupting compounds are found in the environment and include mercury, polychlorinated biphenyls (PCBs), and pesticides such as DDT.

Most of these chemicals (other than mercury) are synthetic, fat-soluble compounds such as pesticides or industrial compounds. The pesticides include chlorinated organic chemicals such as DDT and kepone. Industrial compounds include PCBs, phenols and dioxins, some of which are manufactured intentionally while others are accidental by-products. The most common characteristics of these chemicals are that they are long lived, remaining in the environment or animals for long periods, and dissolve in fats, rather than water. As a result, the DDT and dioxin that was released decades ago still contaminates soils, fish and even humans today.

The sources of these chemicals are twofold: pesticides that get into our food supply and industrial processes that release chemicals that eventually get into our food. Pesticides applied to crops, facilities, animals, animal food, and produce can remain on or in the food. Industrial chemicals can get into our food supply if the emissions or releases are discharged into the environment and ingested by fish and grazing animals. The atmospheric emissions from incinerators and other combustion processes are the major sources of exposure to dioxins and PCBs.

Almost every animal has an endocrine system, which it relies on for normal life functions. In regard to wildlife, hormones regulate various behaviors and processes, including mating behaviors, migration, fat deposition, hibernation, insect metamorphosis, and the shedding of shells by shrimp, crabs and lobster.

Endocrine disruptors are chemicals that interfere with the normal function of hormones and the way hormones control growth, metabolism and bodily functions. There are different ways in which chemicals can interfere with or disrupt hormone function, and there are different kinds of chemicals that disrupt hormones few of these have been tested for carcinogenicity or endocrine disruption.

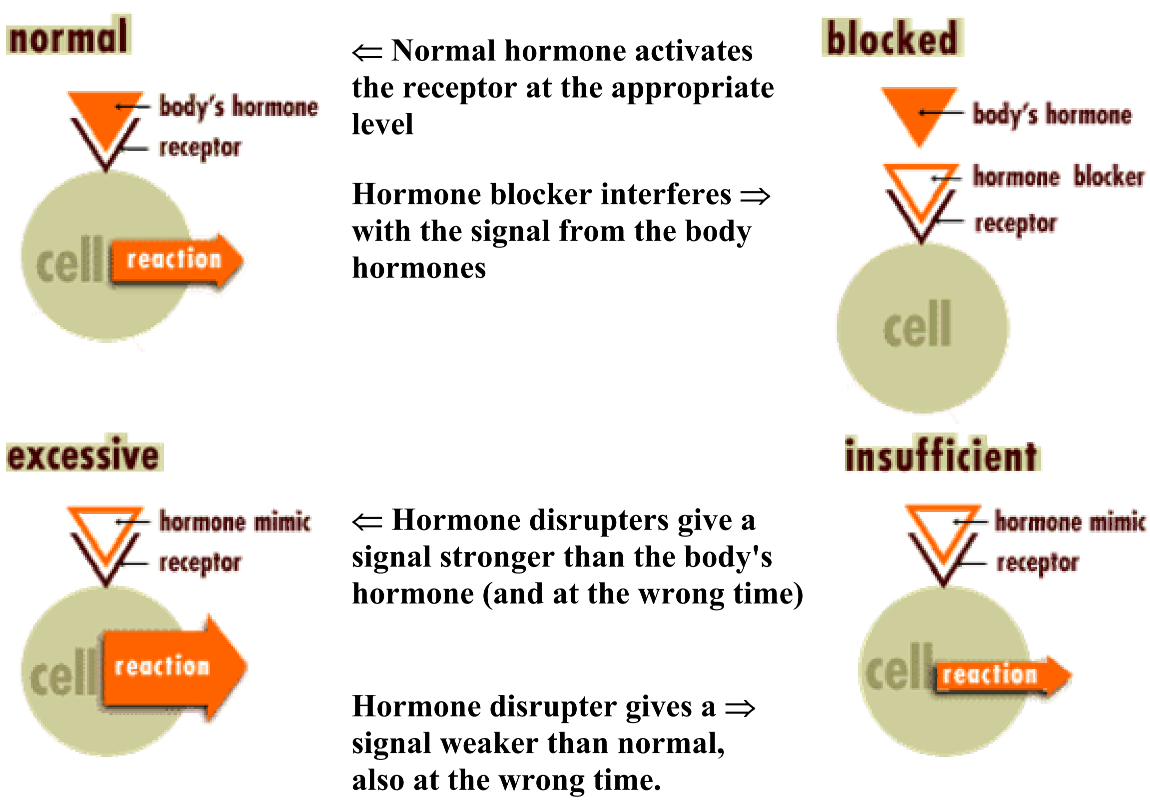
An endocrine system is a hormonal pathway made up of glands that release their products (hormones) into the bloodstream. Hormones are carried through the bloodstream and are responsible for initiating a cellular response in a target cell or tissue. Hormones are active at very low concentrations and most are specific in what responses they cause in the body. Endocrine systems generally control body growth, organ development, metabolism, and regulate body processes such as kidney function, body temperature and calcium regulation.

Hormone disruptors fall into three main categories (Figure 1);
- Hormone mimics are chemicals that act like normal hormones in an animal's body.
- Hormone blockers are groups of disruptors that interfere with the function of naturally occurring hormones. These chemicals often act by binding to the same

protein receptors as natural hormones, preventing the natural hormone from binding to its receptor and further eliminating a response.

- Hormone triggers include chemicals that interfere with normal hormone function by attaching to protein receptors and then trigger an abnormal response or action in the cell. The abnormal action may be growth at the wrong time, an alteration of metabolism, or synthesis of a different product. The best-known type of endocrine disruptor is dioxin, and the dioxin-like chemicals. Dioxin acts through a hormone-like process, but neither mimics nor blocks natural hormones.

Figure 1: Endocrine Disruptor Activity



(*http://whyfiles.org/045env_hormone/main4.html*)

Effects of Estrogenic Substances

Some researchers believe that endocrine-disrupting chemicals are putting many wildlife populations at risk. Exposure to environmental contaminants is believed to be connected to documented problems in wildlife populations, which includes the following:

- Thyroid dysfunction in birds and fish
- Decreased fertility in birds, fish, shellfish, and mammals
- Decreased hatching success in birds, turtles, and fish
- Gross birth deformities in birds, fish, and turtles
- Male fish, birds, and mammals that are feminized

- Female fish, birds, and mammals that are masculinized
- Compromised immune systems in birds and mammals

For fish species living in the Great Lakes, several of these effects are well documented. The Great Lakes contain many synthetic chemicals, including by-products of chemical production (e.g. PCBs and pesticides such as DDT). Salmon between the ages of 2 and 4 have shown a 100% prevalence of thyroid enlargement and bird species that feed on Great Lakes' fish have shown behavior changes, failed reproduction, and early mortality in offspring. In herring gull embryos and newly hatched chicks from Lake Ontario, some males have had oviducts and gonads resembling ovaries, while the oviduct system in female birds can develop abnormally. These findings add to the growing concern of researchers related to the potential effects of environmental estrogens.

- The effects are more commonly found in offspring as opposed to the exposed parent.
- The effect of exposure depends on timing relative to the organism's stage of development.
- The effects of exposure to endocrine-disrupting chemicals may be manifested in entirely different ways in the early embryo, fetus, and newborn as opposed to organisms exposed only in adulthood.
- Finally, because the effects of exposure are often delayed, they may not be fully expressed until the offspring of the exposed adult reaches maturity or even middle age.

Research on wildlife populations, especially in the Great Lakes, has demonstrated that endocrine disrupting chemicals greatly impair animal reproduction and development. Birds with deformed beaks and female birds that nest with other females all have high levels of endocrine disruptors, PCB's, dioxins, and DDE's. Laboratory research reveals that some fish eggs do not develop when exposed to low levels of PCB and dioxin.

Metals

Aquatic organisms may be adversely affected by heavy metals in the environment. The level of toxicity is largely a function of the water chemistry and sediment composition in the surface water system. Slightly elevated metal levels in natural waters may cause the following sublethal effects in aquatic organisms:

- Histological or morphological changes in tissues
- Physiological changes (e.g. suppression of growth and development, poor swimming performance, and changes in circulation)
- Biochemical changes (e.g. enzyme activity and blood chemistry)
- Behavioral changes
- Reproductive changes

Many organisms are able to regulate the metal concentrations in their tissues. Fishes and crustaceans can excrete essential metals, such as copper, zinc, and iron when present in excess and some species can excrete non-essential metals, such as mercury and cadmium, although this is usually less successful.

Research has shown that aquatic plants and bivalves (clams, mussels) are not able to successfully regulate metal uptake; thus, bivalves tend to suffer from metal accumulation in contaminated environments. In estuarine systems, bivalves often serve as indicator organisms in areas of suspected contamination and shell fishing waters are closed if elevated metal levels warrant the issuance of a consumption advisory.

Freshwater studies have shown aquatic plants to be less sensitive to metals than fish and invertebrates; therefore, the water resource is usually managed for the protection of fish and invertebrates since it will ensure aquatic plant survivorship. Metal uptake rates will vary according to the organism and the type of metal. Phytoplankton and zooplankton often assimilate available metals quickly because of their high surface area to volume ratio. The ability of fish and invertebrates to adsorb metals is largely dependent on the physical and chemical characteristics of the metal. With the exception of mercury, little metal bioaccumulation has been observed in aquatic organisms. Metals may enter the systems of aquatic organisms via three main pathways:

- Free metal ions that are absorbed through gills are readily diffused into the blood stream.
- Free metal ions that are adsorbed onto body surfaces are passively diffused into the blood stream.
- Metals that are absorbed onto food and particulates may be ingested, as well as free ions ingested with water.

Ingestion of metals such as lead (Pb), cadmium (Cd), mercury (Hg), arsenic (As), and chromium (Cr^{6+}), may pose great risks to human health. Trace metals such as lead and cadmium will interfere with essential nutrients of similar appearance, such as calcium (Ca^{2+}) and zinc (Zn^{2+}).

Lead is a highly toxic metal that produces a variety of health effects, mainly in young children. There are several ways in which people are exposed to lead, including: deteriorating paint and dust, air, drinking water, food, and contaminated soil. Airborne lead enters the body when you breathe or swallow lead particles or dust once it has settled. Lead can leach into drinking water from lead pipes, copper pipes with lead solder, brass faucets, and can also be found on walls, woodwork, and the outside of your home in the form of lead-based paint. Lead can be deposited on floors, windowsills, eating and playing surfaces, or in the dirt outside the home. Because of size and charge similarities, lead can substitute for calcium and become included in bone; therefore, children are especially susceptible to lead because developing skeletal systems require high calcium levels. Lead that is stored in bone is not harmful, but if high levels of calcium are ingested later, the lead in the bone may be replaced by calcium and mobilized. Once free in the system, lead may cause nephrotoxicity, neurotoxicity, and hypertension.

Fish are most commonly exposed to lead through ingestion of food, water, and sediment containing lead. The lead accumulates in the blood, bones, muscle, and fat of the fish and elevated levels in the water can cause reproductive damage, and blood and neurological changes.

Many ducks and other species of waterfowl consume food from the benthic community of lakes and other bodies of water. In the process of obtaining food from the benthos, these birds may consume small stones and grit that aids in grinding their food. Lead from snagged fishing lures and shot from lead-based shot gun shells formally used (now banned) in waterfowl hunting are sometimes consumed along with grit.

The effect of lead on waterfowl (e.g. loons and swans) has been studied by biologists since the 1970s. This ongoing research has documented that lead sinkers and jigs can account for 10 to 50% of dead adult loons, in loon breeding areas in the Great Lakes region, the northeast United States, and Eastern Canada. Research in New England suggests that mortality in retrieved loons caused by lead ingestion is of greater importance than any other observed mortality factors, including: tumors, trauma, fractures, gun-shot wounds, and infections.

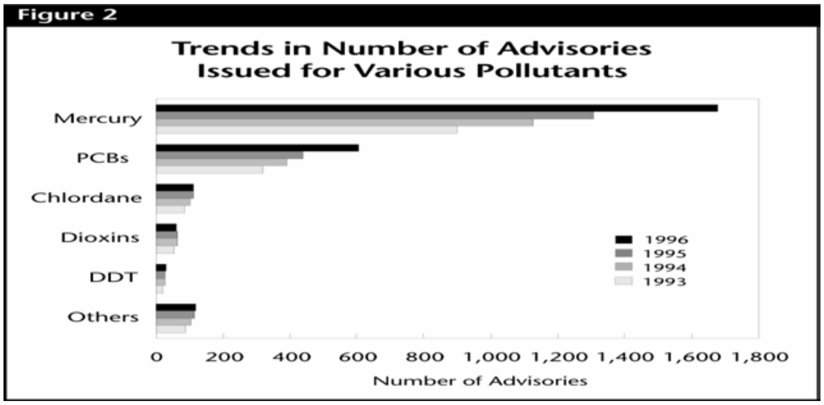
Waterfowl affected by lead poisoning will experience physical and behavioral changes such as loss of balance, gasping, tremors, and impaired ability to fly. Weakened bird species are more vulnerable to predators and may have difficulty feeding, mating, nesting, and caring for their young. Waterfowl exposed to lethal levels of lead become emaciated and often die within two to three weeks after ingesting the lead.

Cadmium may interfere with the metallothionein's ability to regulate zinc and copper concentrations in the body. Metallothionein is a protein that binds to excess essential metals to render them unavailable. When cadmium induces metallothionein activity, it binds to copper and zinc, disrupting the homeostasis levels. Cadmium is used in industrial manufacturing and is a byproduct of the metallurgy of zinc.

Cadmium is found naturally in small quantities in air, water, and soil and because cadmium is a metal, it does not break down and can accumulate over time. Burning of household trash, industrial waste, coal, and oil may release cadmium into the air. In addition, Cadmium can be released from car exhaust, metal processing industries, battery and paint manufacturing, and waste hauling and disposal activities. Once cadmium is in the air, it spreads with the wind and settles onto the ground or surface water as dust. Higher levels of cadmium may be found in soil or water near industrial areas or hazardous waste sites. High levels of cadmium in surface soils usually result from cadmium particles settling from the air. Soils near roads may contain high levels of cadmium from car exhaust. Surface waters also can contain low levels of dissolved cadmium. Cadmium in water tends to sink and accumulate in bottom sediments.

Mercury poses a great risk to humans, especially in the form of methyl-mercury. When mercury enters water it is transformed by microorganisms into methyl-mercury. Symptoms of acute poisoning are pharyngitis, gastroenteritis, vomiting, nephritis, hepatitis, and circulatory collapse. Chronic poisoning is usually a result of industrial exposure or a diet consisting of contaminated fish and may cause liver damage, neural damage, and teratogenesis.

Figure 2: Advisories for Various Pollutants



(*http://www.epa.gov/ost/fish/epafish.pdf*)

Mercury has been well known as an environmental contaminant for several decades. As early as the 1950's it was established that emissions of mercury to the environment could have serious effects on human health. These early studies demonstrated that fish and other wildlife from various ecosystems commonly attain mercury levels of toxicological concern when directly affected by mercury-containing emissions from human-related activities. Human health concerns arise when humans consume fish and wildlife from these ecosystems (Figure 2 and Figure 3).

Figure 3: Fish Consumption Advisories in the U.S. due to Mercury

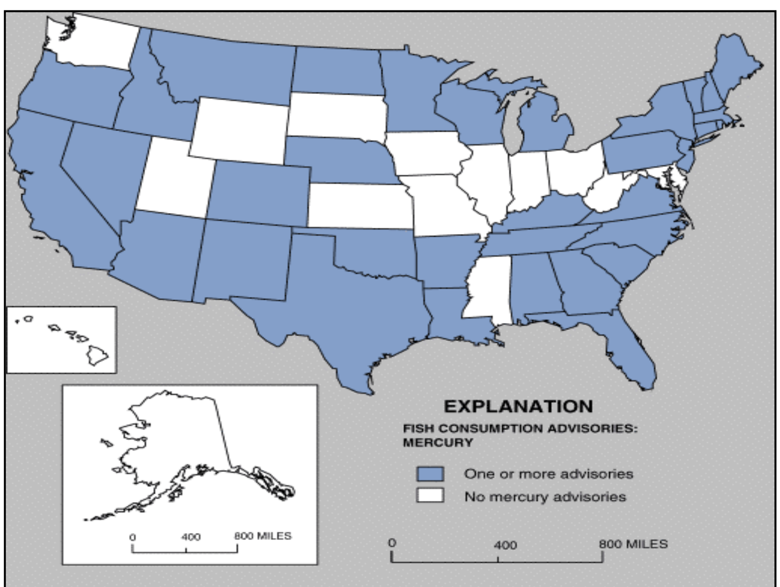


Figure 1. States with at least one fish consumption advisory for mercury. Source: USEPA Fish Consumption Data Base

(*http://water.usgs.gov/wid/FS_216-95.html*)

During the past decade, a new trend has emerged with regard to mercury contamination. Investigations initiated in the late 1980s in the northern-tier states of the United States, Canada, and Nordic countries found that fish, mainly from nutrient-poor lakes and often in very remote areas, commonly have high levels of mercury. More recent fish sampling surveys in other regions of the United States have shown widespread mercury contamination in streams, wetlands, reservoirs, and lakes. To date, 33 states have issued fish consumption advisories because of mercury contamination. In 2001, the EPA issued a national consumption advisory for women who are or may become pregnant, nursing mothers, and young children. EPA recommends that this group consume no more than one meal per week of freshwater fish caught by recreational angling.

The exact mechanism by which mercury enters the food chain remains largely unknown, and probably varies among ecosystems; however, it is known that certain bacteria play an important early role. Studies have shown that bacteria that process sulfate in the environment take up mercury in its inorganic form, and through metabolic processes convert it to methyl-mercury (Figure 4). The conversion of inorganic mercury to methyl-mercury is important for two reasons: methyl-mercury is more toxic than inorganic mercury and organisms require a considerably longer period of time to eliminate methyl-mercury. At this point, higher levels of organisms in the food chain may consume the methyl-mercury-containing bacteria, or the bacteria may release the methyl-mercury to the water where it can quickly be adsorbed by plankton, which are also consumed by higher levels of organisms in the food chain.

Figure 4: Mercury Cycle

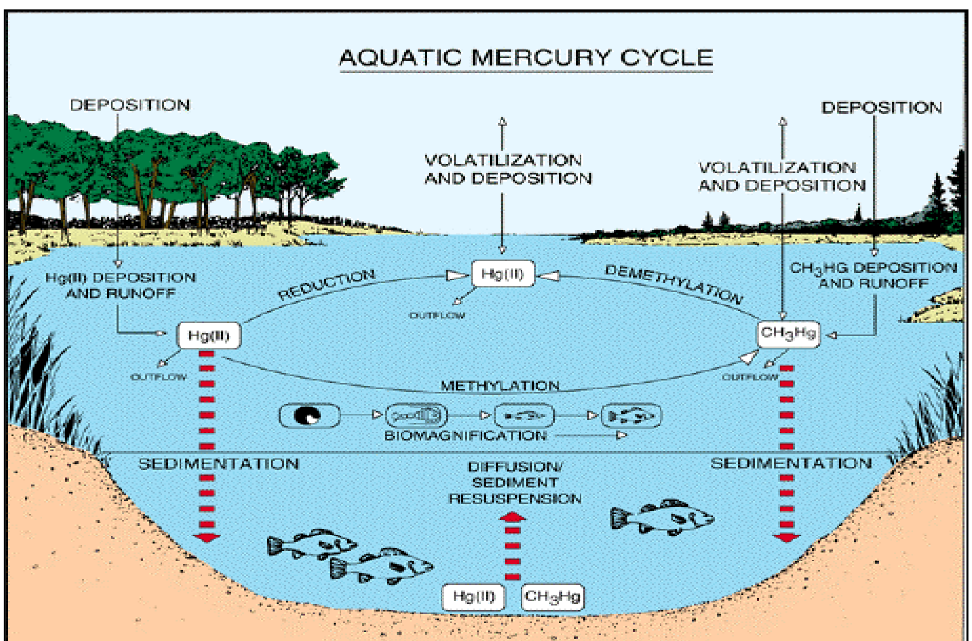


Figure 6. Mercury cycling pathways in aquatic environments are very complex. The various forms of mercury can be converted from one to the next; most important is the conversion to methylmercury (CH_3Hg^+), the most toxic form. Ultimately, mercury ends up in the sediments, fish and wildlife, or evades back to the atmosphere by volatilization. Reprinted with permission from Mercury Pollution: Integration

(*http://water.usgs.gov/wid/FS_216-95.html*)

Mercury can be bioaccumulated by aquatic life. Bioaccumulation is the process by which organisms can take up contaminants more rapidly than their bodies can eliminate them, thus the amount of mercury in their body accumulates over time. If for a period of time an organism does not ingest mercury, its body burden of mercury will decline; however, if an organism continually ingests mercury, its body burden can reach toxic levels. The rate of increase or decline in body burden is specific to each organism. For humans, about half the body burden of mercury can be eliminated in 70 days if no mercury is ingested during that time.

With the exception of isolated cases of known point sources, the ultimate source of mercury to most aquatic ecosystems is deposition from the atmosphere, primarily associated with rainfall. As depicted in the aquatic mercury cycle, atmospheric deposition contains the three principal forms of mercury, although the majority is as inorganic mercury (Hg^{2+}). Once in surface water, mercury enters a complex cycle in which one form can be converted to another and can be brought to the sediments by particle settling, and then later released by diffusion or re-suspension. It can enter the food chain, or it can be released back to the atmosphere by volatilization. The concentration of dissolved organic carbon (DOC) and pH has a strong effect on the ultimate fate of mercury in an ecosystem. Studies have shown that for the same species of fish taken from the same region, increasing the acidity of the water and/or the DOC content generally results in higher body burdens in fish. Many scientists currently think that higher acidity and DOC levels enhance the mobility of mercury in the environment, thus making it more likely to enter the food chain. Many of the details of the aquatic mercury cycle are still unknown and remain an area of active research.

There are many sources of mercury to the environment, both natural and human related. Natural sources include volcanoes, natural mercury deposits, and volatilization from the ocean. The primary human-related sources include: coal combustion, chlorine alkali processing, waste incineration, and metal processing. Human activities have increased the amount of mercury in the atmosphere, and the atmospheric burden is increasing by about 1.5% per year.

Arsenic can cause severe toxicity through ingestion of contaminated food and water. Ingestion causes vomiting, diarrhea, and cardiac abnormalities. Arsenic is an element that is widely distributed in the earth's crust and ordinarily a steel gray metal-like material that sometimes occurs naturally; however, arsenic is usually found in the environment combined with other elements such as oxygen, chlorine, and sulfur. Arsenic combined with these elements is referred to as inorganic arsenic. Arsenic combined with carbon and hydrogen is known as organic arsenic. Understanding the difference between inorganic and organic arsenic is important because the organic forms are usually less harmful than the inorganic forms.

Most inorganic and organic arsenic compounds are white or colorless powders that do not evaporate, they have no smell, and most have no special taste; therefore, the presence of arsenic in food, water, or air is difficult to determine. Inorganic arsenic occurs naturally in soil and in many kinds of rock, especially in minerals and ores that contain copper or lead. When these ores are heated in smelters, most of the arsenic moves up the smokestack and enters the air as a fine dust. Smelters may collect this dust and take out the arsenic as arsenic trioxide; however, arsenic is no longer produced in the United States (all the arsenic the United States uses is imported).

Currently, about 90% of all arsenic produced is used as a preservative for wood to prevent it from rotting and decaying. The preservative most commonly used in known as chromated copper arsenate (CCA) and the treated wood is referred to as "pressure-treated." In the past, arsenic was primarily used as a pesticide, primarily on cotton fields and in orchards. Inorganic arsenic compounds can no longer be used in agriculture; however, organic arsenicals such as cacodylic acid, disodium methylarsenate (DSMA), and monosodium methylarsenate (MSMA) are still used as pesticides, primarily on cotton. Small quantities of arsenic metal are added to other metals, forming metal mixtures or alloys with improved properties. The greatest use of arsenic in alloys is in lead-acid batteries used in automobiles. Another important use of arsenic compounds is in semiconductors and light-emitting diodes.

Arsenic occurs naturally in soil and minerals; therefore, arsenic may enter the air, water, and land from wind-blown dust and may get into water from runoff and leaching. Volcanic eruptions are another source of arsenic. Arsenic is associated with ores mined for metals, such as copper and lead, and may enter the environment during the mining and smelting of these ores. Small amounts of arsenic also may be released into the atmosphere from coal-fired power plants and incinerators because coal and waste products often contain some arsenic.

Arsenic cannot be destroyed in the environment; it can only change forms, or become attached to or separated from particles. Arsenic may change its form by reacting with oxygen or other molecules present in air, water, or soil, or by the action of bacteria that live in soil or sediment. Arsenic released from power plants and other combustion processes is usually attached to very small particles. Arsenic contained in wind-borne soil is generally found in larger particles. These particles settle to the ground or are washed out of the air by rain. Arsenic that is attached to very small particles may stay in the air for many days and travel long distances. Many common arsenic compounds can dissolve in water; therefore, arsenic can get into lakes, rivers, and underground water by dissolving in rain or snow, or through the discharge of industrial wastes. Some of the arsenic will combine with particles in the water or sediment in the benthos of lakes or rivers, and the water will carry some arsenic-contaminated particles through the water column; however, most arsenic ends up in the soil or sediment. Although some fish and shellfish take in arsenic, which can build up in their tissues, most of this arsenic is in a form often called "fish arsenic" that is less harmful.

Chromium is a naturally occurring element found in rocks, animals, plants, soil, and in volcanic dust and gases. The presence of abundant chromium anions in the water is generally a result of industrial waste. The chronic adverse health effects are respiratory and dermatological. Chromium is present in the environment in several different forms. The most common forms are chromium (0), chromium (III), and chromium (VI). No taste or odor is associated with chromium compounds.

- Chromium (III) occurs naturally in the environment and is an essential nutrient.

- Chromium (VI) and chromium (0) are generally produced by industrial processes.
- The metal chromium, which is the chromium (0) form, is used for making steel. Chromium (VI) and chromium (III) are used for chrome plating, dyes and pigments, leather tanning, and wood preserving.

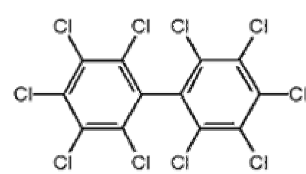
Chromium enters the air, water, and soil mostly in the chromium (III) and chromium (VI) forms. In air, chromium compounds are present mostly as fine dust particles, which eventually settle over land and water. Chromium can attach to soil and only a small amount can dissolve in water and move deeper in the soil to underground water. Fish do not accumulate the majority of chromium in their bodies from water.

Several studies have shown that chromium (VI) compounds can increase the risk of lung cancer. Animal studies have also shown an increased risk of cancer.

- The World Health Organization (WHO) has determined that chromium (VI) is a human carcinogen.
- The Department of Health and Human Services (DHHS) has determined that certain chromium (VI) compounds are known to cause cancer in humans.
- The U.S. EPA has determined that chromium (VI) in air is a human carcinogen. We do not know if exposure to chromium will result in birth defects or other developmental effects in people. Birth defects have been observed in animals exposed to chromium (VI). It is likely that health effects seen in children exposed to high amounts of chromium will be similar to the effects seen in adults (ASTDR, 2001).

Organic Contaminants

Polychlorinated biphenyls, or PCBs, are a group of more than 200 human-made organic chemicals, made up of phenyl and chlorine atoms. A phenyl is a univalent radical with a chemical equation of C_6H_5 (six carbon, five hydrogen atoms) with the symbol Ph.



A biphenyl molecule is comprised of two benzene rings of hydrogen and carbon atoms. Benzene rings serve as the building blocks of petroleum, gasoline, and other fuels. These molecules are extremely flammable. By substituting chlorine for hydrogen atoms, the molecule becomes flame-resistant. Chlorinated biphenyls are any of a group of substances in which chlorine replaces hydrogen. Molecules with more than one chlorine atom are known as polychlorinated biphenyls (PCBs). Polychlorinated biphenyls are toxic and accumulate in animal tissues.

The flame resistance of the polychlorinated biphenyls made them ideal for use in electrical products because they did not burn, break down, or react with other chemicals. Originally produced for use as flame retardants and as electrical insulators in transformers, capacitors, and other electrical equipment, PCBs were used in heating coils, carbonless carbon paper, lubricating oils for industrial drills, caulking compounds for skyscraper windows, electrical motors in refrigerators, in air conditioners, typewriters, power saws, and the like. At one time or another, a wide variety of products including cereal boxes, degreasers, varnishes, lacquers, waterproofing materials, and bread wrappers contained PCBs. The unique properties of PCBs allowed them to be used in the manufacturing of many common products such as plastics, adhesives, paints, and varnishes.

In consistency, PCBs range from light oily fluids to greasy or waxy substances and are clear to yellow in color. During their manufacture and use, PCBs were released into the atmosphere through sewers, smokestacks, weathering of asphalt and other substances containing PCBs, and burning PCB-containing products. PCBs continue to be released from leakage of old equipment, leaching from landfills, and from previously contaminated sediments.

The very characteristic of PCBs that made them wonderful for use in manufacturing makes them problematic in the environment. PCBs are very persistent: they are generally unalterable by microorganisms or by chemical reaction and they do not readily degrade. The stable nature of PCBs also leads to accumulation in the fatty tissues of animals once the PCBs are released into the environment. These accumulations are biomagnified as they work their way through the food web. Because of bioaccumulation, the concentration of PCBs found in fish tissues is expected to be considerably higher than the average concentration of PCBs in the water from which the fish were taken.

Acute toxic effects in the environment include death of animals, birds, or fish, and death or low growth rate in plants. Chronic effects from PCBs may include shortened lifespan, reproductive problems, lower fertility, and changes in appearance or behavior. The primary concern of PCBs in surface water is the chronic effect of bioaccumulation. During the mid-1960s, some environmental scientists began seeing an increase of PCBs in animal tissues. In 1978, the U.S. EPA banned the use of PCBs in all but closed systems of manufacturing. In 1979, the use of PCBs was banned in all applications.

Although PCBs bind strongly to soil and sediments, evaporation to air increases as the amount of moisture, organic carbon, and clay increases in soil and sediment. PCBs tend to accumulate in sediments; therefore, benthic species are most affected by PCB-contaminated sediments. Following the consumption of PCBs by benthic organisms, they tend to bio-accumulate or build up in larger or top-predator fish as they eat the smaller fish or bottom-feeders (benthic organisms). For example, walleye can have higher levels of PCBs than perch because they eat perch and absorb PCBs with each contaminated fish they eat.

Recent studies have shown that exotic species are now playing a major role in food chain transfer of contaminants. The invasion of zebra mussels and round gobies in the Great Lakes has caused increased bioaccumulation of PCBs. Zebra mussels can filter up to one liter of water a day and as a benthic organism, take up PCBs from contaminated sediments. Round gobies heavily prey on zebra mussels and accumulate higher concentrations of PCBs in their tissues. Diet studies of smallmouth bass, rock bass, walleye, yellow perch, and other predatory species have documented that they are now feeding heavily on round gobies; thereby, further accumulating higher concentrations of PCBs. The higher concentrations of PCBs in these sport fish lead to increased health risk to anglers who consume these PCB-containing sport fishes.

Round Goby with Zebra Mussels in its Gut

(Photo by Dave Jude - http://www.sgnis.org/publicat/slide/rg_s7.htm)

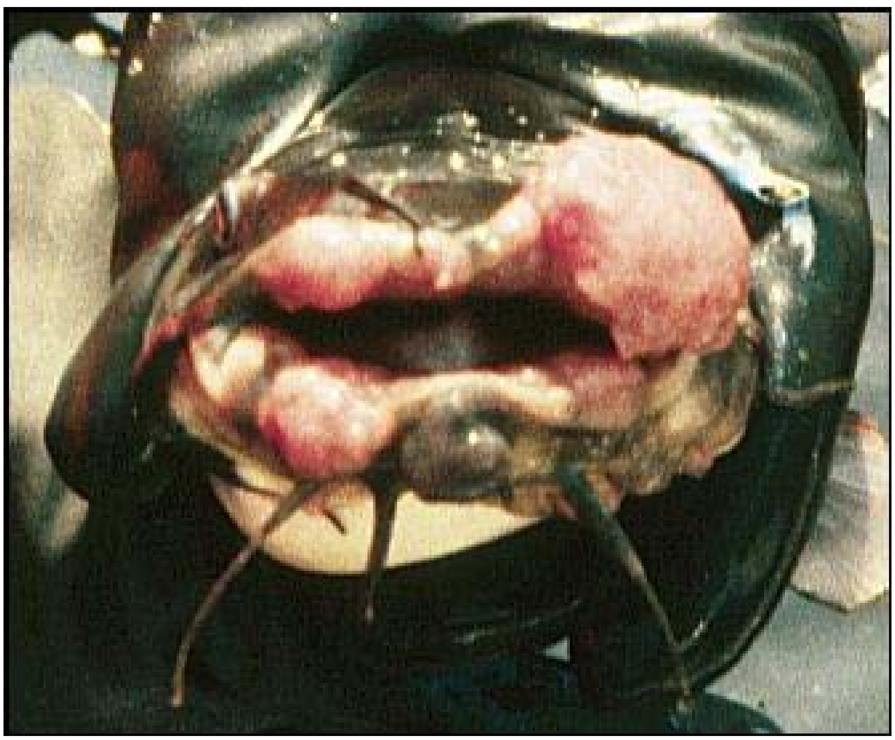
Polycyclic aromatic hydrocarbons (PAHs) are a class of over 100 different organic compounds that are formed during the incomplete combustion of coal, oil, gasoline, garbage, or other organic substances. They are also found in tobacco and charbroiled meat. PAHs are comprised of two or more aromatic rings and are relatively insoluble in water. Because of their insolubility in water, PAHs tend to bind to particles within the water column where they eventually settle into the sediment. Benthic organisms can re-suspend and bioaccumulate PAHs from the sediment, thus making them available to predators higher up the food web.

Some PAHs are manufactured and usually exist as colorless, white, or pale-yellow-green solids. PAHs are found in coal tar, crude oil, creosote, and roofing tar, but a few are used in medicines or to make dyes, plastics, and pesticides. PAHs enter the air most commonly as releases from volcanoes, forest fires, burning coal, and automobile exhaust. PAHs can be found in the air attached to dust particles.

The DHHS has determined that some PAHs may reasonably be expected to be carcinogens. Some humans who have inhaled or touched mixtures of PAHs and other chemicals for long periods of time have developed cancer. Some PAHs have caused cancer in laboratory animals when the animals have inhaled air-containing PAHs (lung cancer), ingested them in food (stomach cancer), or had them applied to their skin (skin cancer).

Animal studies have shown that PAHs can cause harmful effects on the skin, body fluids, and ability to fight disease after both short-and long-term exposure. Bottom feeding fish and crustaceans are likely to ingest PAHs, causing tumors and lesions to the animals. There is currently overwhelming epidemiological evidence (supported by laboratory induction studies) that various tumors discovered in the Great Lakes fishes are caused by carcinogens (chemical contaminants) present in the environment, either acting alone or in the presence of other tumor promoters. Brown bullheads painted with PAH contaminated sediment from the Buffalo River developed skin cancer after two years at an incidence of 38%.

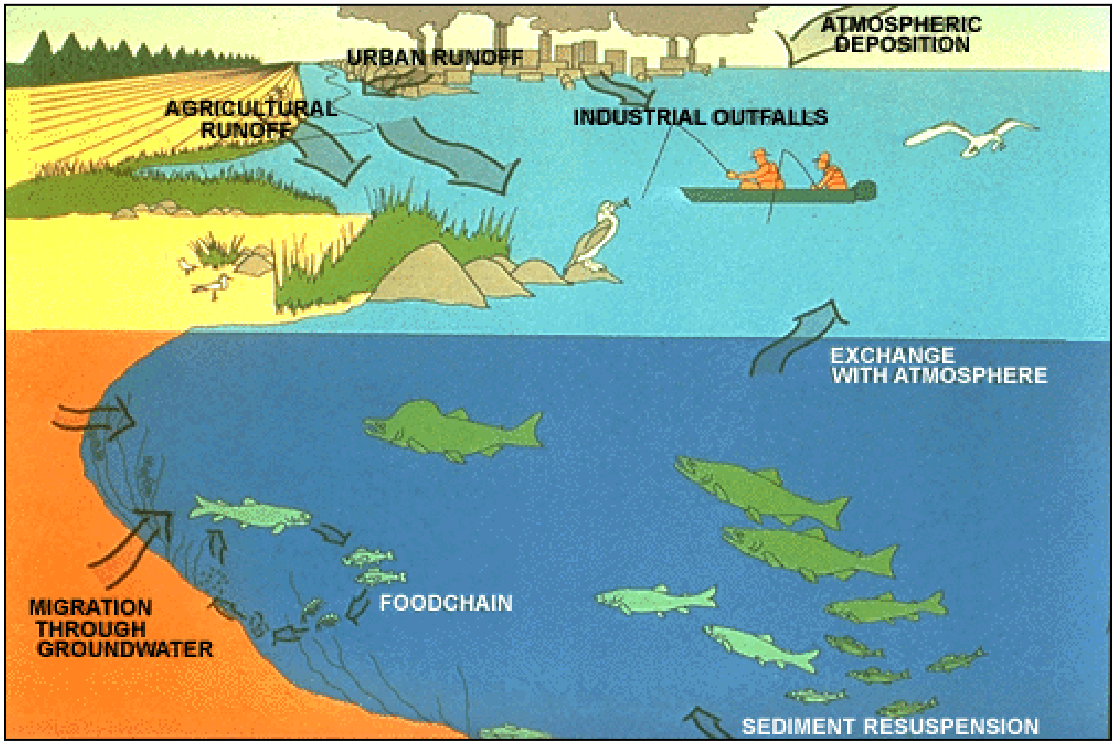
Brown Bullhead with Orocutaneous Tumors



(*Photo provided by Eric Obert of Pennsylvania Sea Grant*)

Some PAH particles can readily evaporate into the air from soil or surface waters and can degrade by reacting with sunlight and other chemicals in the air, over a period of days to weeks. PAHs enter water through discharges from industrial and wastewater treatment plants. Microorganisms can break down PAHs in the soil or water after a period of weeks to months. In soils, PAHs are most likely to bind tightly to particles; certain PAHs move through soil to contaminate underground water. PAH contents of plants and animals may be higher than the PAH contents of soil or water in which they live.

Cycling of PAHs and Other Contaminants



(*http://www.glerl.noaa.gov/pubs/brochures/wcontflyer/wcont.html*)

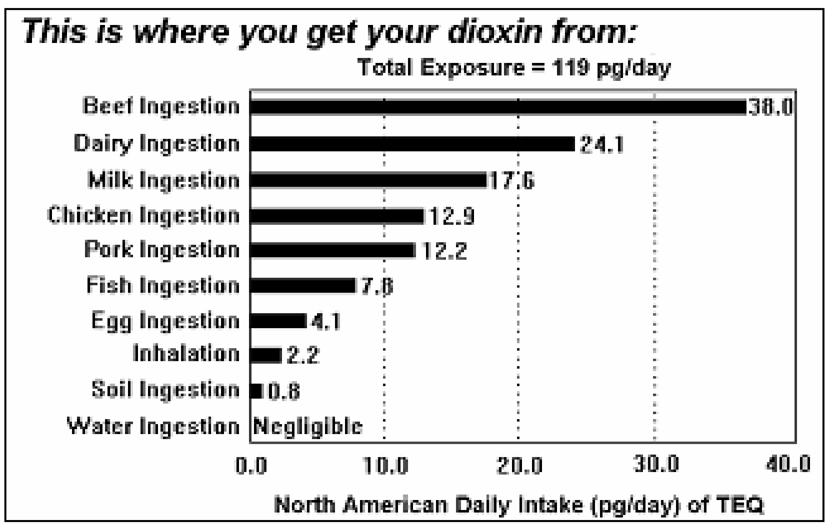
Dioxin is found in herbicides (e.g. agent orange) and is released from municipal incinerators and paper mills. Dioxin is listed as a toxic man-made chemical. Furans and PCBs are very similar to dioxin in structure but are 100 times less toxic. Sometimes these three compounds - dioxins, furans and PCBs are referred to collectively as "dioxin." They are scored for their dioxin-like potency, and toxic equivalency factor (TEF) relative to dioxin. The other non-dioxin-like PCBs have different toxic properties.

The boiling point of PCBs is about 325-366 °C, so PCBs can tolerate fairly high temperatures without exploding or burning. Unfortunately, furans are created from PCBs at 250-450 °C, which means that simply heating PCBs can convert them to create additional furans in the mixture.

It is possible that high-temperature pan-frying or grilling of fish or other PCB contaminated foods could convert some of the PCBs to more-toxic dioxins and furans. PCBs will burn at higher temperatures; however, some of the PCBs will be converted to furans and dioxins, especially in poorly controlled, inconsistent fires. When PCBs are burned at very high temperatures, as in hazardous waste incinerators designed for this purpose, the PCBs may be completely broken down, but as the gases leave the smoke stack they cool and recombine to form new PCBs, furans and dioxins. Municipal waste incineration is the major global source of dioxins. Over time, aging PCB mixtures oxidized, especially if exposed to heat or other catalyzing chemicals. PCB oils used in transformers often included chemicals designed to inhibit oxidation of the oils.

The higher chlorinated and dioxin-like PCBs, and dioxins and furans are much more likely to be absorbed or ingested and retained in fish and in the human body, in comparison to the lower-chlorinated, lightweight PCBs. These dioxin-like compounds are also slower to breakdown. The lightweight PCBs are more likely to degrade, blow off in the air or stay dissolved in the water. This means that even in cases where total PCB measurements in fish are declining, the more toxic dioxin-like forms could be increasing locally in intensity and health effects.

Dioxin has been the subject of an intensive health reassessment by the EPA and scientists for the past 10 years. They have determined that dioxins and dioxin-like compounds such as PCBs are known carcinogens. They suspect that the average American may face a 1 in 1,000 risk of cancer due to these compounds, perhaps a risk as high as 1 in 100, because contamination has become so widespread in our general food supply (particularly in meat, dairy, poultry, fish, eggs and other fatty animal products).



(*http://www.ejnet.org/dioxin/*)

Furans are produced unintentionally from many of the same processes that produce dioxins and also during the production of PCBs and Furans have also been detected in emissions from waste incinerators and automobiles. Furans are structurally similar to dioxins and share many of their toxic effects. There are 135 types of furans and their toxicity varies. Furans persist in the environment for long periods and are classified as possible human carcinogens. Food, particularly animal products, is the major source of exposure for humans, and furans have also been detected in breast-fed infants.

Atrazine is a white, crystalline solid organic compound. It is a widely used herbicide for control of broadleaf and grassy weeds. Atrazine was estimated to be the most heavily used herbicide in the United States in 1987 through 1989, with its most extensive use for corn and soybeans in the midwest. Atrazine has been greatly restricted since 1993.

Atrazine may be broken down in soil and water through microbial activity and other chemicals, particularly in alkaline conditions; however, atrazine does not evaporate. Atrazine can bind to some soils but generally tends to escape into groundwater. Atrazine has staying power and may persist in soil and water for over a year, and traces have been found in water supplies, and fruits, vegetables, meat, and dairy products. Atrazine is also an endocrine disrupting toxin. In studies looking at the effect of ten different endocrine disrupting chemicals on estrogen function, only DDT had a more severe effect than atrazine.

Chemical degradation of atrazine may be more important environmentally than biodegradation. Atrazine may hydrolyze fairly rapidly in either acidic or basic environments, yet is fairly resistant to hydrolysis at neutral pH. Atrazine may maintain a very high to medium mobility class in soils and should not be strongly absorbed by sediments. Atrazine is not expected to bioaccumulate or volatilize.

Chlordane was used in the past to control pests on crops (corn, grapes, strawberries, etc.), control fleas and ticks, and mange on pets. Chlordane is a probable human carcinogen, and the United States banned most uses of chlordane in 1978 and all uses except termite control in 1987. Canada discontinued use in 1990.

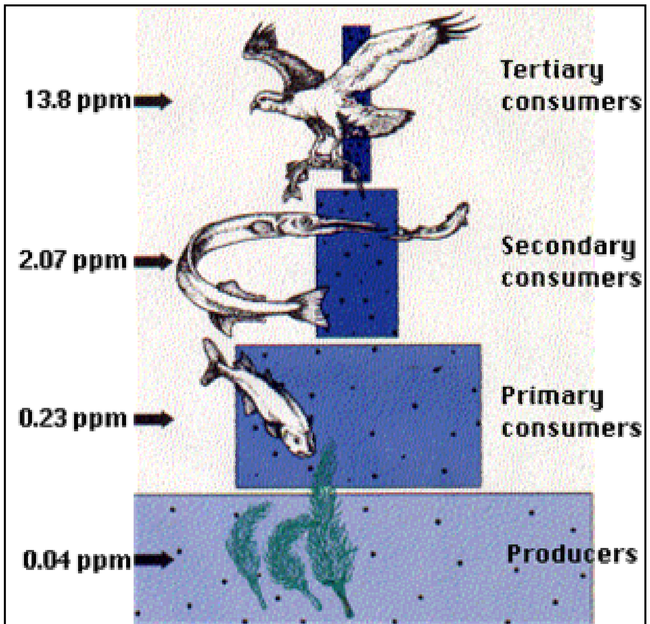
Dichlorodiphenyltrichloroethane (DDT) and metabolites was used in large quantities in the 1950s and 1960s on cotton fields, orchards and other crops, and in unsuccessful extermination campaigns against the Japanese beetle, spruce budworm, gypsy moth, and Dutch elm disease bark beetle. DDT was also an ingredient in the pesticide Kelthante (Dicofol). DDT breaks down into toxic metabolites, primarily DDE. DDT and DDE are probable human carcinogens and endocrine disrupters. The United States banned DDT (except for public health emergencies) in 1973, and banned Kelthane (unless it contained less than 0.1% DDT) in 1988. Canada discontinued DDT in 1985. DDT is still used in other countries, mostly for insect control.

A major known source of DDT in the Lake Huron watershed is in the Pine River located near St. Louis, Michigan at a chemical processing plant that has been shut down. Remediation of contaminated sediment was initiated at the site in the spring of 1999, and some DDT has been stockpiled in the Lake Huron Basin and shows up when agricultural pesticides are collected. DDT has been detected in Lake Huron water, fish, and wildlife.

The development of the pesticide DDT in the 1940s was considered a major breakthrough in the battle against diseases such as malaria, and in controlling crop pests. DDT is highly effective and inexpensive to produce, and was the most widely used pesticide in North America and other countries from 1946 to 1972. Agricultural use of DDT has since been banned in North America following the discovery of DDT and its breakdown products ability to cause widespread reproductive failures in eagles and other wildlife species. The bald eagle was listed as an endangered species in 1967. As DDT accumulated in individual bald eagles from their natural feeding habitats, the species' reproduction drastically decreased. This was due to DDT breakdown products impairing calcium release necessary for eggshell formation. This resulted in thin eggshells and the reproductive failures. Although DDT continues to be used in other parts of the world, levels of DDT in the North American environment have decreased significantly since this pesticide was banned, and species impacted by DDT such as the bald eagle are recovering.

DDT and its metabolites are identified as Lakewide Management Plan critical contaminants because they are responsible for wildlife consumption advisories and are identified as potential contaminants for bald eagles once they re-establish their shoreline nesting territories.

Bioaccumulation of DDT in an Aquatic Ecosystem



The numbers are representative values of the concentration in the tissues of **DDT** and its derivatives (in parts per million, ppm)

(*http://depts.washington.edu/envir202/Lessons/Lesson19.pdf*)

The upper Great Lakes are the largest source of DDT and its metabolites to the Lake Ontario basin (96 kg/year). Atmospheric deposition and sources within the Lake Ontario basin contribute approximately 33.5 kg/year combined. Much of the tributary loadings likely consist of atmospheric fallout in the watershed given the banning of these materials from use in the watershed. Approximately 143 kg/year of DDT leave Lake Ontario (141 kg/year) and the St. Lawrence River (2 kg/year) via volatilization to the atmosphere.

Dieldrin and aldrin were widely used pesticides from the 1950s until 1970 for crops like corn and cotton. Sunlight and bacteria help convert aldrin to dieldrin; therefore, dieldrin is more commonly found in the environment. Pure aldrin and dieldrin are white powders with a mild chemical odor; less pure commercial powders have a tan color. Neither substance occurs naturally in the environment. Because of concerns about damage to the environment and potentially to human health, the EPA banned all uses of aldrin and dieldrin in 1974, except to control termites. In 1987, EPA banned all uses. Aldrin and dieldrin bind tightly to soil and slowly evaporate to the air. Dieldrin, once in the soil and water, breaks down very slowly. Plants take in and store aldrin, at which time it is rapidly converted to dieldrin. In animals, dieldrin is stored in the fat and exits the body very slowly.

Dieldrin is identified as a Lakewide Management Plan critical contaminant in Lake Ontario because dieldrin concentrations in water and fish tissue exceed the U.S. Great Lakes Water Quality Initiative (GLI) criteria throughout the lake. The GLI criterion for water is 0.0000065 parts per billion (ppb) and Lake Ontario water averages 0.17ppb. The corresponding GLI fish tissue criterion is 0.0025 parts per million (ppm) and most of Lake Ontario's fish clearly exceed this criterion as dieldrin is detectable at concentrations ranging from approximately 0.005 to 0.030 ppm.

Although the GLI criteria are being exceeded, dieldrin concentrations in the environment have been steadily declining. Between 1985 and 1995, dieldrin concentrations in Lake Ontario have declined from 0.35 to 0.17 ppb based on information collected through Niagara River and Wolfe Island monitoring programs. The upper Great Lakes are the largest source of dieldrin to the Lake Ontario basin (43 kg/yr). Atmospheric deposition and point and nonpoint sources of dieldrin within the Lake Ontario basin are approximately equal (13 kg/year and 9 kg/year).

Fire Retardants

Mirex was found in elevated levels in Lake Ontario fish during the 1960s, triggering lakewide fish consumption advisories. Mirex is a white crystalline solid and is odorless, and has not been manufactured or used in the United States since 1978. Mirex was used in the past to control fire ants and as a flame retardant in plastics, rubber, paint, paper, and electrical goods from 1959 to 1972. Investigations determined that most of the mirex originated from a chemical production facility on the Niagara River. Use and production of mirex, also known as dechlorane and chlordecone, is now banned in North America. Mirex is identified as a Lakewide Management Plan critical contaminant because levels in some Lake Ontario fish continue to exceed human health standards; a number of fish consumption advisories exist. Although mirex is most widely known for its use as a pesticide, approximately 75 percent of the mirex produced was used as a flame retardant in a variety of industrial, manufacturing, and military applications.

Available sales records suggest that more than 50,000 pounds of mirex were used for industrial and manufacturing flame retardant purposes in the Lake Ontario basin. More than 75,000 pounds of mirex were used as a flame retardant in other Great Lakes basins. Most of the mirex entering Lake Ontario originates in the Niagara River basin (1.8 kg/year) and an additional 0.9 kg/year enters via the Oswego River. Approximately 0.7 kg/year of mirex leaves Lake Ontario via the St. Lawrence River. No reliable estimates of atmospheric deposition or volatilization are available at this time (Great Lakes Commission, 2002). Mirex degrades slowly in the environment, and may persist for years in soil and water. It does not evaporate to any great extent from surface water or surface soil and does not dissolve easily in water, but it commonly binds to soil and sediment particles. Mirex is not likely to travel far through the soil and into underground water; however, mirex can accumulate in fish or other organisms that live in contaminated water or preys on other contaminated animals. Animal studies have shown that ingesting high levels of mirex can harm the stomach, intestine, liver, kidneys, eyes, thyroid, and nervous and reproductive systems (ATSDR, 2002).

Polybrominated biphenyls (PBBs) and **Polybrominated diphenyl ethers** (PBDEs) are manufactured chemicals found in plastics used in a variety of consumer products ranging from computer monitors, televisions, textiles, and plastic foams to make them difficult to burn. Because they are mixed into plastics rather than bound to them, they can dissipate from the plastic and find their way into the environment. PBBs and PBDEs are similar, but not identical compounds; both are colorless to off-white solids, and are mixtures of up to 209 individual component chemicals called congeners. PBBs enter the air, water, and soil during their manufacture and use, and small amounts entered the environment from improper incineration of plastics containing PBBs (ATSDR, 2002).

In 1973, commercial flame retardant containing PBBs was accidentally mixed into feed for dairy cattle, livestock, and poultry in the state of Michigan. The feed was widely used and eventually lead to the widespread PBB-contamination of milk, meat, and eggs, and poisoning in animals. Over 9 million people were exposed to PBBs from food. Due to the widespread exposure, research was conducted to understand better the toxicology of PBBs, poisoned animals, and exposed humans. The effects of PBBs were found to be essentially the same as those seen for PCBs (Swedish EPA, 2000). Most of what we know about the health effects of PPBs in people comes from studies related to people in Michigan who consumed PBB-contaminated animal products for several months. Some residents complained of nausea, abdominal pain, loss of appetite, joint pain, fatigue, and weakness; however, it could not be clearly established that PBBs were the cause of these health problems. There is stronger evidence that PBBs may have caused skin problems, such as acne, in some people who consumed contaminated food. Some workers exposed to PBBs via inhalation and skin contact for days to months also developed acne.

Studies in animals exposed to large amounts of PBBs for a short time or to smaller amounts for longer time show that PBBs can cause weight loss, skin disorders, nervous and immune systems effects, and effects on the liver, kidneys, and thyroid gland (ATSDR, 2002).

PBDEs are added to plastic and furniture foam as "flame retardants" to suppress fire. The chemical structures of the PBDEs are similar to those of other chemical families, including the PCBs and the dioxins; however, while the PCBs and many of the dioxins have chlorine in their chemical structures, PBDEs are made with bromine, a close chemical relative to chlorine. Like dioxins and PCBs, PBDEs are long lasting in the environment and some of the PBDEs accumulate in the wax of plants and the fat in animals; therefore, they accumulate in the food chain. PBDEs are persistent bioaccumulative chemicals, which can enter the womb and concentrate in breast milk, causing the transfer of contamination from one generation to the next (CBE, 2000). PBDEs enter air, water, and soil during their manufacture and use in consumer products. In air, PBDEs can be present as particles, but eventually settles into soil or water. Sunlight can degrade some PBDEs in air; however, PBDEs do not dissolve easily in water, rather they bind to particles and settle to the bottom of rivers or lakes. Some PBDEs can accumulate in fish.

There is no definite information on health effects of PBDEs in humans. Rats and mice that consumed food with moderate amounts of PBDEs for a few days had effects on the thyroid gland. Those that consumed smaller amounts for weeks or months had effects on the thyroid and the liver. Preliminary evidence suggests that PBDEs may cause neurobehavioral alterations and affect the immune system in animals (ATSDR, 2002).

Fish Consumption Advisories

Fish are a lean low-calorie source of protein and can play an integral part of a healthy diet; however, some of the fish species anglers catch from the Great Lakes and surrounding watersheds may contain chemicals that could pose health risks.

(*Photo provided by Eric Obert of Pennsylvania Sea Grant*)

Many chemical contaminants are present in surface waters at very low concentrations. Some of these chemicals can bioaccumulate in aquatic organisms via their diet and become concentrated at levels that are much higher than in the water itself. This is especially true for substances that do not break down readily in the environment, like persistent chemicals such as PCBs.

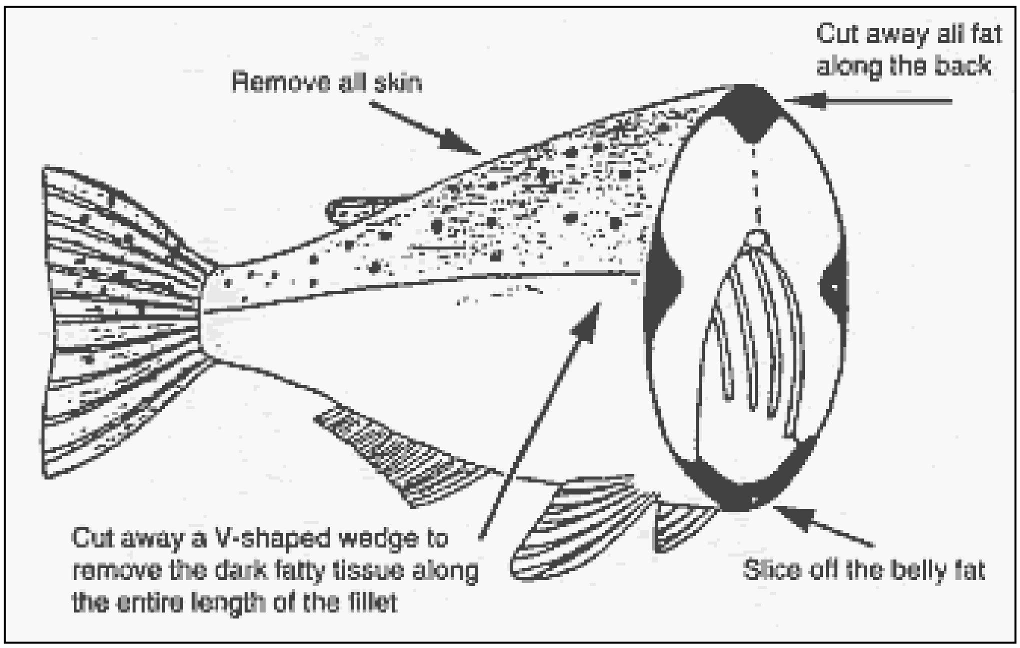
Small fish and zooplankton eat large quantities of phytoplankton. In doing so, any toxic chemicals accumulated by the phytoplankton are further concentrated in the bodies of the organisms that ingest them. This is repeated at each hierarchy of the food chain. The concentration of some chemicals in the tissues of top predators, such as lake trout and large salmon, can be millions of times greater than the concentration in the water. Although bioaccumulative chemicals are present in other food, the concentration that accumulates in fish due to the complex hierarchy of the food chain of fish, are much higher than in other food.

Certain chemicals, such as PCBs and DDT, accumulate in the bodies of fish over time because they are excreted very slowly. Children and women of childbearing age will retain ingested chemicals for long periods of time, and newborns and unborn children may be exposed to harmful chemicals if the mother has regularly consumed contaminated fish in the years before her pregnancy. During pregnancy and nursing, it is unwise for the mother to eat even small amounts of contaminated fish.

Two chemicals of concern, mercury and PCBs, can be passed onto the baby during pregnancy and breast-feeding. Because of their size, small children and babies cannot tolerate exposure to harmful chemicals in the same respect as an adult. These chemicals can cause problems with the growth, behavior and development of young children, and may contribute to other health concerns in adults. People most at risk from eating contaminated fish are:

- Pregnant and Nursing Mothers
- Women of Child-Bearing Age
- Young children and Babies
- People who regularly eat fish from contaminated areas.

Proper Trimming of Fish Helps Reduce PCB Consumption



(*http://www.pserie.psu.edu/seagrant/communication/pamphlet/pamphlet2*.html*l*)

** ***<u>Please note that Mercury is concentrated in the muscle tissue and can not be removed by cutting away the fatty portions.</u>***

Risk Assessments

All foods, including fish, contain environmental contaminants. When setting the acceptable level of a contaminant in commercial fish, federal governments take into account several factors in addition to potential health effects including: assumptions about the amount of human fish consumption, the species consumed, where the fish come from, and economic considerations. State and provincial governments provide information to consumers regarding consumption of sport-caught fish. This information is not regulatory, it is meant for use as guidance, or advice. Some states use the federal commercial-fish guidelines for the acceptable level of contaminants when giving advice for eating sport caught fish; however, consumption advice offered by most agencies is based on human health risk. This approach involves interpretation of studies of health effects from exposure to contaminants. Each state or province is responsible for developing fish advisories for protecting the public from pollutants in fish and tailoring this advice to meet the health needs of its citizens. As a result, the advice from state and provincial programs is sometimes different for the same lake and species within that lake.

(*http://www.epa.gov/glnpo/aoc/ashtabula.html*)

The toxic endpoints used in risk assessments for calculating safe fish consumption levels are subtle (the effects are not easily recognizable or attributable to a particular exposure and that exposure does not cause immediate harm). Numbness of fingertips, dizziness, and the sensory loss that might occur from toxic exposures to methyl-mercury, might also easily be attributed to the normal aging process. Developmental problems resulting from *in utero* exposure to PCBs are difficult to measure or even separate from confounding factors like smoking or alcohol consumption. The variability in response of individuals exposed to persistent bioaccumulative toxic (PBT) chemicals dictates a more conservative approach, perhaps producing guidance that is over protective of a large portion of the population.

As the data from ongoing PCB studies are published, the weight of evidence for PCB effects on neurodevelopment is growing. In particular, studies in Taiwan (Chen et al. 1992), Michigan (Jacobson et al. 1985, 1990a; Jacobson and Jacobson 1996), Oswego, New York (Darvill et al. 2000), The Netherlands (Patandin et al. 1999), Germany (Walkowiak et al. 2001; Winneke et al. 1998), and the Faroe Islands (Budtz-Jorgensen et al. 1999; Grandjean et al. 2001) have now all reported negative associations between prenatal PCB exposure and measures of cognitive functioning in infancy or childhood. Only one published study in North Carolina has failed to find any association between PCB exposure and cognitive outcomes (Gladen and Rogan 1991). It is particularly noteworthy that the levels of exposure in some of the more recent studies, the Oswego cohort, for example, are significantly lower than in the earlier studies, yet negative impacts on cognitive functioning are still being reported.

It is important that people are aware of contaminants in fish and the actions that can be taken to reduce exposure particularly those people who are at greatest risk from overexposure to contaminants found in fish. Exposure to detrimental levels of environmental contaminants can cause a variety of negative health effects. The precise level of contaminant exposure that is detrimental to an individual is going to vary with his/her age, sex, genetics, current physical condition, and previous exposure of that individual. Individuals within a population will vary in their sensitivities to environmental contaminants. It is not possible to determine a priori of which individuals within a population are going to be most sensitive to contaminant exposure, and because governments need to protect sensitive individuals in the population, the advice governments provide may be over protective for some portion of the population. The table provided below illustrates the differences between the fish advisories developed for carp, channel catfish, chinook salmon, and lake trout. Differences among fish advisories do not only occur among the different Great Lakes, but they also occur within different States and Provinces bordering the Great Lakes.

Fish Advisories for the Great Lakes and Lake Champlain: Carp, Channel Catfish, Chinook Salmon & Lake Trout

	Carp	Channel Catfish	Chinook Salmon	Lake Trout
Lake Michigan				
Illinois	DO NOT EAT	DO NOT EAT	1 meal/month fish under 30"	1 meal/month fish under 23"
			1 meal/2months fish over 30"	Do not eat fish over 27"
Indiana	1 meal/month	DO NOT EAT	1meal/month fish under 26"	1 meal/month fish under 21"
			Do not eat fish over 30"	Do not eat fish over 26"
Michigan	DO NOT EAT	DO NOT EAT	1 meal/month fish under 26"	Do not eat fish over 26"
			1 meal/2months fish over 26"	
Wisconsin	No advisory	No advisory	1 meal/month fish under 30"	1 meal/month fish under 23"
			1 meal/2months fish over 30"	Do not eat fish over 23"
Lake Superior				
Michigan	No advisory	No advisory	1 meal/month	1 meal/week fish 18-30"
				Do not eat fish over 30"
Minnesota	No advisory	No advisory	1 meal/week fish under 22"	1 meal/week fish under 21"
				1meal/month fish 21-34"
Wisconsin	No advisory	No advisory	1 meal/week fish under 22"	1 meal/week fish under 22"
			1 meal/month fish over 22"	1 meal/month fish 21-34"
Lake Huron				
Michigan	DO NOT EAT	1 meal/week fish under 18"	1 meal/month	1 meal/week fish 10-22"
		Do not eat fish over 18"		Do not eat fish over 22"
Lake Erie				
Michigan	DO NOT EAT	DO NOT EAT	1 meal/month	1 meal/2 months
New York	1 meal/week	1 meal/week	1 meal/weed fish under 19"	1 meal/week
Ohio	1 meal/month	1meal/2months fish under 16"	1 meal/month fish over 19"	2 meals/month
Pennsylvania	1 meal/month fish under 20"	1 meal/2 months	No advisory	1 meal/2 months
	Do not eat fish over 20"			
Lake Ontario				
New York	Do not eat fish over 25"	Do not eat fish over 25"	Do not eat fish over 20"	1 meal/month fish under 25"
				Do not eat fish over 25"
Lake Champlain				
New York	No advisory	No advisory	No advisory	1 meal/month fish over 25"
Vermont	No advisory	No advisory	No advisory	3 meals/month

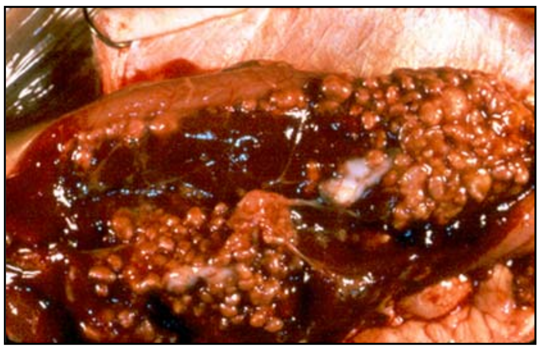
(*This table represents an example of how fish consumption advisories vary among Great Lakes and among states bordering the Great Lakes*)

While the average person in the Great Lakes basin may not be at risk of experiencing adverse health effects from exposure to contaminants through the consumption of fish, there are some people who are at risk. These include people who eat a lot of Great Lakes fish, regularly eat large predator fish, eat fish from highly contaminated waters, or eat a large amount of fish over a short period of time. In addition, the developing fetus and young children are at greater risk than adults.

Fish Tumors

The purpose in assessing fish tumors and deformities is to use these as an indicator of environmental degradation of the aquatic ecosystem and a measure of health impairment to fish populations. Tumors are defined as heritably altered, independent (meaning functions outside host), relatively atypical tissue growths. Tumors can be induced genetically, virally and chemically. Deformities are defined as twisted, missing, forked, or bulging body parts including deformed fins, barbels, abdomen or skeleton. Deformities are caused by several factors, including: environmental degradation (i.e. chemical contaminants), rapid temperature change during early development, viruses, bacteria, parasites, and/or fish spawning and migration. Lesions are open sores, exposed tissue, and/or prominent bloody areas.

Internal Tumors



External Tumors

(*Photos provided by Dr. John Gannon as part of his presentation at the 2003 Fish Tumors Related to Great Lakes Areas of Concern Conference held in Erie, PA*)

Various contaminant analysis programs in recent years have concluded that the Great Lakes environment is receiving and accumulating various organic and inorganic contaminants. Some of these contaminants, acting alone or with other contaminants, are thought to be carcinogens. Fish tumor induction data, tumors in wild fish populations, and the presence of contaminants (i.e. carcinogens) have provided evidence that chemical carcinogenesis is occurring in the Great Lakes and its watersheds.

Chemically induced cancer, in humans, was first investigated in 1775 by Dr. Percival Pott, who discovered the occurrence of scrotal cancers in chimneysweepers who were exposed to soot. The idea of chemical induction of tumors in wild fish population was reported in early literature; however, the concept was slow to develop. In the 1930s the first carcinogen was identified to be benzo(a)pyrene (PAH). In 1962, almost a decade later, the first epidermal papillomas (tumors) were discovered in brown bullheads in Deep Creek Lake, Maryland. The Maryland Fisheries Department discovered that some of the white suckers and bullheads in this lake had liver neoplasms. The discovery of tumors in these two species led to the recommendation of the use of these benthic fish as "indicators of environmental contaminants (carcinogens)."

Dawe first advanced the hypothesis that neoplasms in wild fish might be caused by environmental carcinogens in 1964. Zebrafish were exposed to diphenyl nitrosamine in a lab study conducted in 1964, resulting in the development of cancer in the zebrafish. In 1977, Dr. Sonstegard's study included data on the prevalence of papilloma in white sucker. Dr. Sonstegard hypothesized that both a virus and environmental carcinogens were involved in the etiology of this neoplastic disease. In 1990, Dr. Harshbarger and Dr. Clark listed fifteen tumor epizootics that were reported in fish from the Great Lakes and surrounding watersheds. Today, research is still being conducted in hope of better understanding the link between contaminants in the environment and tumors in various fish species.

Great Lakes Areas of Concern With Confirmed Tumor or Other Deformity Presence

(*http://www.epa.gov/glnpo/aoc/*)

The terms tumor, neoplasm and cancer are often used interchangeably. Cancer is used to describe a malignant disease in which the cancer (unless treated) has the capacity to invade and spread throughout the body, leading to the death of the host species. Neoplasia is abnormal and uncontrolled growth that often produces a tumor (neoplasm) that may or may-not be cancerous.

Role of Contaminants in Fish Tumor Induction

There is currently overwhelming epidemiological evidence (supported by laboratory induction studies) that various tumors discovered in the Great Lakes fishes are caused by carcinogens (chemical contaminants) present in the environment, either acting alone or in the presence of other tumor promoters. Brown bullheads painted with polycyclic aromatic hydrocarbons (PAH)-contaminated sediment from the Buffalo River developed papilloma after two years at an incidence of 38%. An oral papilloma incidence rate over 70% was discovered in black bullheads in a waste water treatment facility in 1979 and 1980, and following a 90% reduction in the residual chlorine in the pond by 1983, papilloma rates decreased by 23%.

For tumor formation to occur, several chemicals require the need for metabolic activity before they can induce neoplasia. As part of the detoxification and excretion process, some chemicals (e.g. benzo(a)pyrene) are converted to polar metabolites by enzymes in the liver.

These enzymes are highly reactive and interact with critical components of the cell and initiate the cancer process. Since the liver provides the essential source of detoxification enzymes, it is frequently the site of cancer in fish that have been exposed to chemical contaminants (carcinogens) in the lab.

A wide variety of contaminants (i.e. carcinogens), including PAHs, benzo(a)pyrene (BaP) and dimethylbenzanthracene, induce liver cancers in fish. PAHs have a flat, hydrophobic stage, which makes them hard to excrete from the body. The shape of PAHs allow them to insert themselves into DNA, forming a PAH contaminated-DNA adduct, where they interfere with the proper function of the DNA. PAHs are very un-reactive, and in the liver they are oxidized and these oxidized PAHs react with the DNA to form the DNA adducts. Research conducted by Dunn in 1987 detected aromatic carcinogen-DNA adducts in the livers of brown bullheads collected from PAH-contaminated sites in the Great Lakes. DNA adducts can be characterized as chemical contaminants covalently bound to DNA. The formation of these adducts is potentially significant in carcinogenesis because the DNA adduct can give rise to chromosomal aberrations, DNA strand breaks, oncogene activation, and tumor suppressor gene activation, which can lead to cancer formation.

Virally Induced Fish Tumors

Not all fish tumors are caused by chemical contaminants. Viruses or viral-chemical combination action can cause many benign and malignant tumors in Great Lakes fish. Lymphosarcoma is an infectious disease of viral origin. It commonly affects Muskellunge and Northern Pike. The disease usually starts as small tumor below the skin and can develop into tumors the size of a tennis ball. The tumors often appear as soft whitish-gray, red or pink growths; however, upon maturity they may rupture and appear as dead tissue. Lymphosarcoma can be transferred from fish to fish through contact during spawning, and tumor prevalence and development are highest in late winter and spring and lowest in the summer (suggesting a role of temperature in tumor development). Lymphosarcoma is thought to be a virally induced cancer because a viral enzyme is often detected, the occurrence of the disease is in uncontaminated environments, disease patterns are suggestive of an infectious process, laboratory based transmission studies are positive, and the outbreaks appear to be seasonally based. Other tumors believed to be virally induced include lymphocystis and dermal sarcomas in walleye, papillomas in carp, and possibly lip and body papillomas in white suckers.

Parasitic infestation can also lead to health problems in fish. *Heterosporis*, a newly found microsporidian parasite, has infected walleye and yellow perch in Lake Ontario, and inland lakes of Wisconsin and Minnesota. The parasite is more closely related to fungi than other spore forming parasites, and it causes the flesh of the fish to become opaque and destroys the value of the fish. The effect of water temperature on the development of *Heterosporis* has not yet been studied; however, most microsporidian parasites to not develop under 15° Celsius. Also, at this time it is unknown whether birds and mammals that consume fish infected with *Heterosporis* can pass viable spores through their feces. A major concern is the possibility of the spores suspended in the water column being transferred to other bodies of water via boats.

Lymphocystis: A Viral Disease on Walleye

(*Photo provided by Eric Obert of Pennsylvania Sea Grant*)

Fish Tumors in Great Lakes Areas of Concern

The International Joint Commission (IJC) characterized fish tumors and other deformities as beneficial use impairments. One of fourteen use impairments, this beneficial use impairment (BUI) is used by Areas of Concern (AOCs) as criteria for the listing and de-listing process. Current criteria pertaining to fish tumors and other deformities include:

1) An intestinal or liver tumor prevalence of $\geq$ 5 to 7% occurs in common native near shore species of benthic dwelling fish (brown bullhead), walleye, perch or salmonid species offshore. Samples must consists of 30 fish, each of which is 250 mm or greater in length. Tumors are defined as neoplasms of either intestinal, bile duct, or liver cells as determined by histopathology
2) A prevalence of lip tumors $\geq$ 8-10% or overall external tumors $\geq$ 13-15% in white sucker and brown bullhead. Tumors are defined as papillomas or other neoplasms as determined by histopathology. Samples must consist of at least 30 fish, each of which is 250 mm in length or greater.
3) A Deformities, Erosion, Lesions & Tumors (DELTs) external anatomy index of > 0.5% occurs. (Baumann; LaMP, 2000)

Brown Bullhead with Several Tumors

(*Photo provided by Eric Obert of Pennsylvania Sea Grant*)

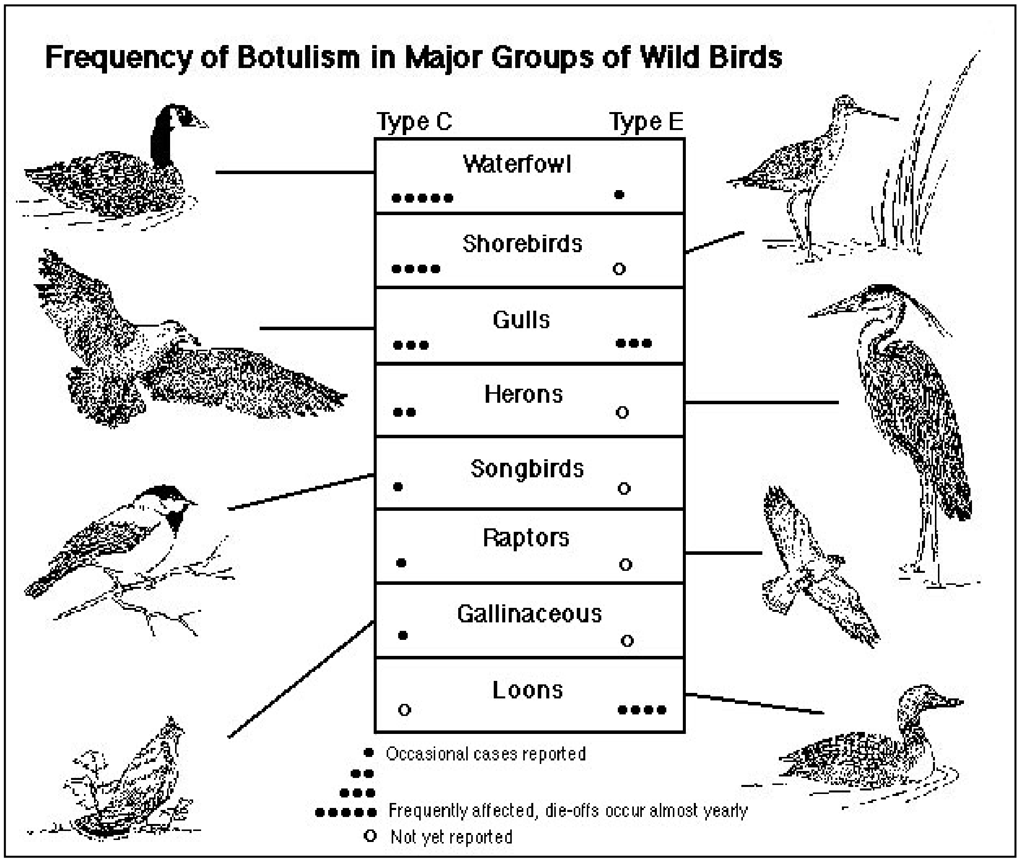
Botulism

Botulism, a disease caused by *Clostridium boulinum*, has been recognized as a major cause of mortality in migratory birds dating back to the early 1900s. Although type C botulism has caused the die-off of thousands of waterfowl (especially ducks) across the western United States, type E has been mainly restricted to fish-eating birds in the Great Lakes. Other outbreaks of type E have sporadically occurred in Alaska, Florida, and California, with periodic outbreaks occurring in 2000, a large die-off of waterfowl occurred in Lake Erie and type E botulism was isolated in these outbreaks. In 2001, a large die-off of benthic fishes like sheepshead occurred along the shores, followed in the fall by another die-off of fish-eating birds. The botulism outbreak continued in Lake Erie in 2002 with major mortality events involving the kill of gulls, common loons and long tailed ducks. In addition, small botulism outbreaks were reported in Lake Huron and Lake Ontario.

(*Bob Wellington of PA Erie County Health Department with dead sturgeon*)

The bacterium is classified into seven types (A-G) by using characteristics of the neurotoxins that are produced. The toxins produced by *C. botulinum* are among the most potent biological poisons, warranting human health and safety concerns. These neurotoxins bind to the receptors on nerve endings, impacting neuromuscular function, which results in the paralytic effect or paralysis, and respiratory impairment. Because of the paralysis, birds often drown before dying from the respiratory impairment.

Although type C and type E avian botulism outbreaks occurred in the Great Lakes in the past, there are some significant differences between the two types. Type C botulism primarily impacts dabbling ducks and bottom-feeding waterfowl, although shorebirds may also fall victim to this type of botulism. In type C botulism, the bacterium, *C. botulinum*, does not produce toxin unless a specific "phage" or virus infects it. This relationship with a phage is not known to exist with type E. Type E botulism typically impacts fish-eating birds like loons and grebes.



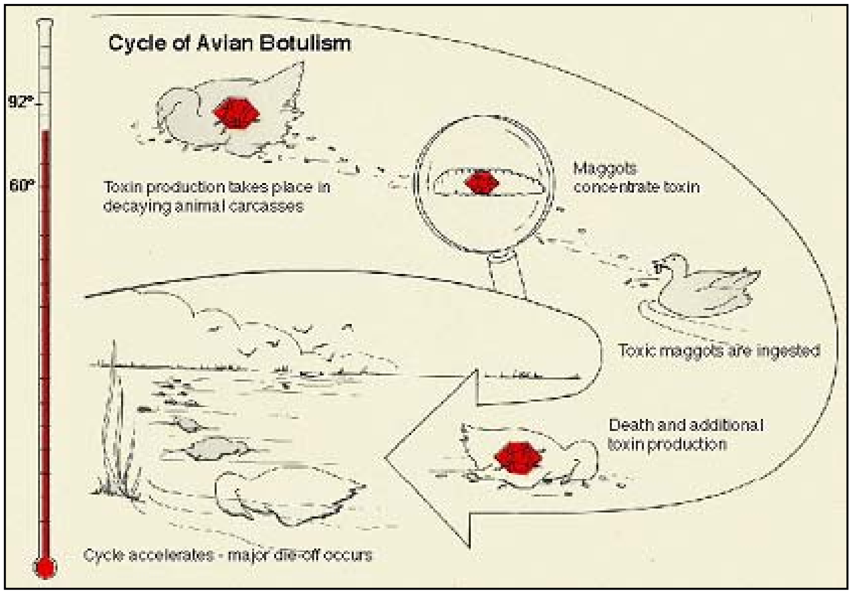
(*http://www.nwhc.usgs.gov/facts/avian.html*)

Spores of both type C and type E botulism are naturally found in anaerobic habitats such as soils and aquatic sediments, and can also be found in the intestinal tracts of live, healthy animals. The spores can remain in the ecosystem for extended periods of time, even years, and are quite resistant to temperature extremes and drying. In the absence of oxygen, with a suitable nutrient source, and under favorable temperatures and pH, spores can germinate and production of vegetative bacterial cells can occur.

Botulism toxin is only produced during vegetative growth, not when the bacterium is in its spore stage. Decaying animal and insect carcasses provide favorable conditions for botulism toxin production since the decay process uses up oxygen and creates anaerobic conditions.

It has long been known that type C botulism is perpetuated through a carcass-maggot cycle. Researchers have now determined that type E botulism can also spread through this cycle. Birds and fish that die from botulism will decay and become hosts for maggots. The maggots may contain the botulism toxin and if fed upon by birds, the cycle is continued.

Cycle of Avian Botulism



(*http://www.nwhc.usgs.gov/facts/avian.html*)

Several species of gulls that are common in the Great Lakes region have been impacted by type C and type E botulism. Type E toxin has been found in carcasses of several species of Great Lakes fish, including smallmouth bass, freshwater drum (sheepshead) and round gobies. In addition, quagga mussels found in long tailed ducks have tested positive for type E botulism. Researchers are currently studying the role that round goby and quagga mussels may be playing as vectors of the disease in the outbreaks on the Great Lakes.


(*Photographs provided by Larry Smith of the Pennsylvania Game Commission*)

Human Health Considerations

Human botulism is typically caused by eating improperly canned or stored foods and normally involves type A or type B botulism toxin. There were several fatalities during the 1960s in the Great Lakes basin attributed to type E toxin, but these were caused by eating improperly smoked or cooked fish that contained the toxin. Botulism poisoning can be avoided by following a few simple steps pertaining to harvesting and preparing fish and waterfowl.

Proper cooking of fish and waterfowl will kill bacteria found in food items. When canning or smoking fish or waterfowl, methods should be used that incorporate sufficient heat to insure that any toxins will be killed off. Anglers and hunters should avoid harvesting any floating, sick or dying fish or waterfowl, or those demonstrating unusual behavior, in areas where avian botulism has occurred. When cleaning your fish, try to avoid contact with the gut contents and your knife.

Proper disposal of dead fish and birds from beach areas may prevent increased wildlife mortality through the maggot botulism cycle. People should not handle dead birds or fish with bare hands. The use of gloves or an inverted plastic bag is recommended in order to avoid risks. If a diseased or dead bird is handled without gloves, hands should be thoroughly washed with hot soapy water.

In case of a die-off, individuals are urged to contact local agencies responsible for fish and wildlife management to notify them of fish and bird mortalities. It is important to record the location, type of birds or fishes, and number of carcasses found. Anglers and hunters should follow agency recommendations in handling dead fish and wildlife. In certain areas, burying of the carcasses is allowed; in other areas incineration may be recommended. If birds are to be collected, they should be placed in heavy plastic bags to avoid the spread of botulism-containing maggots.

Links for Great Lakes and Lake Champlain Fish Consumption Advisories

Illinois
http://www.idph.state.il.us/envhealth/fishadv/fishadvisory02.htm

Indiana
http://www.state.in.us/isdh/dataandstats/fish/fish_2001/fish_cvr_2001.htm

Michigan
http://www.michigan.gov/mdch/1,1607,7-132-2944-13110--,00.html

New York
http://www.health.state.ny.us/nysdoh/environ/fish.htm

Ohio
http://www.epa.state.oh.us/dsw/fishadvisory/index.html

Pennsylvania
http://www.fish.state.pa.us/

Wisconsin
http://www.dnr.state.wi.us/org/water/fhp/fish/advisories/

Illinois, Indiana, Michigan, Minnesota, New York, Ohio, Pennsylvania & Wisconsin
http://www.iet.msu.edu/regs/state.htm

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http://www.lcbp.org/fishadvs.htm

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Ashtabula River Area of Concern < http://www.epa.gov/glnpo/aoc/ashtabula.html>

Avian Botulism <http://www.nwhc.usgs.gov/facts/avian.html>

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World Resources Institute.
<http://www.wri.org/health/estrogen.html>